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NUTRIENT INTAKE AND GASTRIC CANCER IN MEXICO

Lizbeth LÓPEZ-CARRILLO[1]*, Malaquías LÓPEZ-CERVANTES[1], Mary H. WARD[2], Jaime BRAVO-ALVARADO[1] and Armando RAMÍREZ-ESPITIA[1]

[1]*National Institute of Public Health, Cuernavaca, Mexico*
[2]*Occupational Epidemiology Branch, National Cancer Institute, NIH, Bethesda, MD, USA*

In contrast to the decreasing trends observed in most countries, gastric-cancer mortality has remained at about the same level in Mexico throughout the last 40 years. As part of a study carried out in the metropolitan area of Mexico City, an assessment of nutrient intake and gastric cancer is presented here. The study population comprised 220 cases of gastric cancer and 752 population-based controls. Our results showed 70 to 80% reduction in the risk of developing this tumor, associated with the intake of polyunsaturated fat, fiber and vitamin E; and this effect was independent of the histological type of the tumor (i.e., intestinal or diffuse). On the other hand, an increased risk of gastric cancer was related to the consumption of saturated fat ($OR_{Q4vs.Q1}$ = 4.37, 95% CI 1.89–10.12) and, cholesterol ($OR_{Q4vs.Q1}$ = 2.39, 95% CI 1.23–4.64), but such effects were restricted to the intestinal type of gastric cancer. In the whole study population, mono-unsaturated fat intake increased the risk for gastric cancer, and a marginally significant increasing trend was observed for protein consumption. The findings from this study add information about the role of specific nutrients in the etiology of gastric cancer. *Int. J. Cancer* 83:601–605, 1999.

The role of nutrient intake on gastric-cancer (GC) risk has been thoroughly investigated in North America, Asia and Europe (Kono and Hirohata, 1996). In most countries, a decrease in the incidence of this tumor has been reported over the last 4 decades; in contrast, GC mortality in Mexico did not follow the same trend (López-Carrillo *et al.,* 1997). Nevertheless, the GC mortality rate is considerably lower in Mexico than that reported for other countries (World Health Organization 1996, 1997) (Japan 38.7, Spain 17.3, Mexico 5.2, per 100,000 inhabitants). In spite of previous statements, GC is currently the second most important cause of death due to malignant tumors in Mexico (Secretaria de Salud de México, 1992), and no information is available regarding the role of nutrient intake and the incidence of such tumors in the country.

The ability to evaluate nutrient intake depends on having comprehensive food-composition tables and the technical capacity (software) for summarizing individual consumption, not only from the standpoint of particular food items, but also from dishes prepared according to local recipes. The absence of these elements partly explains why epidemiological information on nutrient intake and health outcomes is scarce in Mexico and in other Latin American countries.

Here we report on the relationship of nutrient intake and gastric-cancer incidence, using data from a population-based case-control study carried out in the metropolitan area of Mexico City. An evaluation of the effect of chili-pepper consumption has been published (López-Carrillo *et al.,* 1994).

MATERIAL AND METHODS

The data-collection period for the study spanned from early 1989 to the end of 1990, and comprised the whole metropolitan area of Mexico City. The study population included 220 histologically confirmed, newly diagnosed patients with gastric cancer and a representative sample of 752 controls, who were residents of the same area.

All the cases were identified at 15 Mexico City hospitals, and represented altogether about 80% of the total number of stomach-cancer cases reported every year to the National Cancer Registry. The diagnosis of each patient was histologically confirmed by a single pathologist, according to the Laurèn classification (Laurèn, 1965). For the study, we accumulated information on 82.3% of the 267 eligible cases (all individuals newly diagnosed with gastric cancer who were at least 20 years old and lived in the Mexico City metropolitan area), identified within the space-time limits set for the study.

Approximately 3 controls for each case were selected and matched following the expected distribution for gender and age (5-year groups) in the case group. The control group was assembled by taking a representative sample of residents of the same area, identified from a listing of 1,375 blocks of households that, in turn, was drawn from the master sampling frame created for the system of national-health surveys. One household was randomly selected within each block, and a 5-year age interval was assigned at random to each house. Whenever more than one eligible control (healthy individuals, 20 years of age and older and residents of the Mexico City metropolitan area) lived in the household, only one was chosen at random to be interviewed. For this group, the response rate reached 95.1%, that is, 752 out of 790 eligible controls. More detailed information on the recruitment procedures is reported elsewhere (López-Carrillo *et al.,* 1994).

Structured interviews were carried out by trained personnel, to obtain information about dietary patterns, socio-demographic and clinical characteristics, for the 12-month period before the onset of symptoms, for the cases (abdominal pain, vomiting, dysphagia and/or post-prandial fullness) and the 12 months prior to interview, for the controls. Although the interviewers were aware of the participant case-control status, they were blind about the potential role of specific nutrients in regard to gastric-cancer risk.

Dietary information

Data on 70 food items was obtained using a standardized food frequency questionnaire (Hernández-Avila *et al.,* 1998), which includes information on dairy products, fruits, meats, processed meats, fish, vegetables, sodas and candies. For each item, a pre-determined portion was considered, which, in turn, was converted into grams according to the standard portions defined by the software Food Intake Analysis System (FIAS) 3.0 (University of Texas, Houston Health Science Center, School of Public Health, Human Nutrition Center, United States Department of Agriculture, Agricultural Research Service, 1996). The options for frequency of consumption included 10 categories, from ''never'' to ''6 times per day.'' To calculate the frequency of consumption of each food item, we used the midpoint of the consumption category, and whenever appropriate we adjusted the intake for seasonality.

Nutrient intake

The individual intake of 13 *a priori* selected nutrients, and the total calories from foods and beverages were estimated by the FIAS software. The advantage of this package is that it contains

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*Correspondence to: Av. Universidad 655, Col. Sta. Ma. Ahuacatitlan, Cuernavaca, Morelos, CP 62508, Mexico. Fax: (5273) 11-23-38.
E-mail: lizbeth@insp3.insp.mx

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nutritional information about many foods currently consumed in Mexico, since it was developed in a state that has a large population of Mexican origin, which has kept in its diet the traditional foods and dishes of its ancestors.

The nutritional values used by the software FIAS 3.0 were compared with the corresponding ones, as reported by the Mexican food composition tables (MFCT) developed by the Mexico National Institute of Nutrition (Instituto Nacional de la Nutrición Salvador Zubirán, 1996). Whenever the nutrient values between FIAS and the MFCT differed by more than 10%, the values from the latter were used. We added nutritional values for some local foods and dishes not contained in the FIAS 3.0 dataset.

Due to lack of information about the amounts of different vitamins and other nutrients in the many types of chili peppers consumed in Mexico, such food items were excluded from the estimated total nutrient intake; instead, we controlled for chili-pepper consumption in the statistical models.

Statistical analysis

Nutrient intake was categorized on the basis of the quartile cut-off points corresponding to the controls. By fitting unconditional logistic-regression models, we estimated the odds ratios (OR) for nutrient intake and gastric cancer, including terms for age, gender, total calories, chili-pepper consumption, socio-economical status, cigarette smoking, salt consumption, history of peptic ulcer and type, duration and place of the interview. The analysis was also performed in separate steps for the intestinal and diffuse histological types of gastric cancer (Laurèn, 1965).

In order to test for linear trends, we treated the ordinal nutrient intake variable as continuous. In this step, we considered p values of 0.05 or less as evidence for a dose-response relationship. All analyses were carried out with the STATA version 4.0 software.

RESULTS

Selected characteristics of the study population are shown in Table I. Due to the age-matching procedure, mean ages for cases and controls were similar and around 58 (range 20–98 years). There was a significant excess of women in the control group (60.64% *vs.* 44.55%); in contrast, cigarette smoking and salt consumption were significantly higher for cases. The distribution of socio-economical status was similar in both groups.

Table II shows the mean daily nutrient intakes for the gastric-cancer cases and the controls according to gender. Overall, the cases reported significantly lower mean daily intakes of polyunsaturated fat and fiber, regardless of their gender. Vitamin-E consumption was also significantly lower among cases, but this difference was significant only for the females. For males and females combined, the cases had a significantly higher cholesterol intake than the controls.

TABLE I – SELECTED CHARACTERISTICS OF THE STUDY POPULATION

Characteristic	Cases (n = 220)	Controls (n = 752)
Age (years)		
Average	57.18	59.19
Min.–Max.	24–88	20–98
Gender (%)*		
Females	44.55	60.64
Males	55.45	39.36
Cigarette smoking (%)*		
Non-smokers	39.09	53.19
Ever-smoked	60.91	46.81
Salt consumption*,[1]		
Never	38.18	62.37
Sometimes	61.82	37.63
Socio-economic status		
Low	34.69	29.84
Medium	39.29	46.77
High	26.02	23.39

*$p < 0.05$.–[1]Adding salt after tasting the food.

The multivariate-adjusted OR for the quartile distributions of the macronutrients (Table III) showed a significant positive linear trend for the risk of gastric cancer with increasing consumption of saturated fat. The OR for the highest quartile compared with the lowest quartile of consumption frequency were significantly elevated for saturated fat (OR, 4.37; 95% CI, 1.89–10.12); monounsaturated fat (OR, 3.45; 95% CI, 1.39–8.56); and cholesterol (OR, 2.39; 95% CI, 1.23–4.64). Likewise, an increasing risk of gastric cancer corresponded to an increasing pattern of protein consumption ($OR_{Q4VS.Q1}$, 4.13; 95% CI, 1.56–10.94), with a marginally significant test for trend ($p = 0.063$). A statistically significant inverse linear trend was also observed for frequent consumption (Q4 *vs.* Q1) of polyunsaturated fat (OR, 0.20; 95% CI, 0.09–0.46), and fiber (OR, 0.22; 95% CI, 0.09–0.55). However, the statistical significance of the linear trends for saturated fat and cholesterol remained only for the intestinal type of gastric cancer, although the OR were also elevated for cases of the diffuse type. The protective effect due to polyunsaturated fat and fiber intake was present regardless of the histological type of the tumor.

The results of the micronutrient-intake analyses (Table IV) uncovered a protective effect of vitamin E, with a significant linear trend that held for the 2 histological types of the tumor (highest *vs.* lowest quartile, 0.36; 95% CI, 0.16–0.82). Similarly, a protective effect was seen for the highest category of folate consumption among cases of the intestinal type of gastric cancer (OR, 0.70; 95% CI, 0.22–2.23). In this data set, we found no evidence for a relationship of GC with intake of vitamin A (retinol equivalents), carotenes or vitamin C.

DISCUSSION

The findings of this study confirm that several dietary factors identified elsewhere are also relevant for explaining gastric-cancer incidence in Mexico, despite the fact that gastric cancer has not declined in Mexico during the last 40 years. As reported by other studies, we found that the 2 histologic types of gastric cancer (intestinal and diffuse) are related to the intake of specific nutrients.

The results pertaining to an increased risk of gastric cancer due to the consumption of saturated fat and cholesterol are similar to those reported by González *et al.* (1994) in Spain. In both studies, the relationship between fat intake and gastric cancer was stronger for the intestinal type of GC. Nitrites and salt contained in processed and smoked meats are thought to play a role in the etiology of GC (World Cancer Research Fund and American Institute for Cancer Research, 1997). In this regard, it is possible that our findings on saturated fat, cholesterol, and protein consumption should be considered as proxy indicators for processed-meat consumption. We also found an increased risk for monounsaturated fat, the major sources of which are poultry, meat and milk in the Mexican diet (Instituto Nacional de la Nutrición Salvador Zubirán, 1996), suggesting that, in general, a high consumption of animal products may be a risk factor in our population.

Vegetables and fruit are the main sources of fiber, vitamin E and folate (World Cancer Research Fund and American Institute for Cancer Research, 1997). Epidemiologic studies, mainly using a case-control design, have consistently shown that consumption of vegetables and fruit protects against gastric cancer (Kono and Hirohata, 1996; World Cancer Research Fund, and American Institute for Cancer Research, 1997; Steinmetz and Potter, 1991). We found a strong protective effect of fiber and vitamin-E consumption, which remained significant regardless of the histological type of the tumor, and a moderate protective effect of folate intake, which was observed only for the intestinal type of gastric cancer.

The effect of dietary fiber has been evaluated by 5 case-control studies (González *et al.*, 1994; Risch *et al.*, 1985; Buiatti *et al.*,

TABLE II – MEAN DAILY NUTRIENT INTAKE IN THE STUDY POPULATION

Nutrient/day	Males		Females		All	
	Cases (n = 122)	Controls (n = 298)	Cases (n = 98)	Controls (n = 456)	Cases (n = 220)	Controls (n = 752)
Calories (Kcal)	2,112.52	2,083.78	1,999.80	2,004.00	2,062.22	2,035.40
Macronutrients						
Protein (g)	70.51	71.07	69.87	69.28	70.22	69.98
Fat (g)	71.49	73.91	71.54	72.25	71.52	72.91
Saturated fat (g)	21.35	20.19	21.68	20.76	21.50	20.54
Mono-unsaturated fat (g)	22.88	23.02	22.82	22.28	22.77	22.57
Polyunsaturated fat (g)	21.10	24.38*	21.13	23.10*	21.11	23.60*
Cholesterol (mg)	313.62	294.91	301.96	280.36	308.43	286.10*
Carbohydrates (g)	297.99	292.45	279.41	281.41	285.76	289.72
Fiber (g)	24.67	27.20*	24.15	26.93*	24.44	27.04*
Micronutrients						
Vitamin A (Ret eq.)	3,003.28	3,039.48	2,957.46	2,934.28	2,982.87	2,975.88
Carotene (Ret eq.)	1,899.77	1,816.37	1,759.51	1,826.18	1,837.30	1,822.32
Vitamin C (mg)	345.91	307.11	314.55	306.84	331.94	306.95
Vitamin E mg (alfatocoferol eq.)	15.17	16.78*	15.21	16.29	15.19	16.49*
Folate (μg)	356.25	382.97	352.34	374.04	354.51	377.58

*$p < 0.05$.

TABLE III – ADJUSTED ODDS RATIOS (OR)[1] FOR THE EFFECT OF MACRONUTRIENT INTAKE ON GASTRIC CANCER

Nutrient/day	All adenocarcinomas				Intestinal (n = 98)			Diffuse (n = 95)		
	Cases	Controls	OR	95% CI	Cases	OR	95% CI	Cases	OR	95% CI
Protein (g)										
≤52.25	51	188	1.00	—	20	1.00	—	27	1.00	—
52.26–65.81	47	188	1.74	(0.93–3.25)	17	1.50	(0.59–3.81)	20	1.00	(0.43–2.30)
65.81–84.47	64	188	3.15	(1.55–6.38)	34	4.50	(1.70–11.95)	24	1.64	(0.62–4.36)
≥84.48	58	188	4.13	(1.56–10.94)	27	4.40	(1.12–17.38)	24	2.54	(0.66–9.78)
p value for trend			0.063			0.169			0.520	
Fat (g)										
≤54.12	59	188	1.00	—	21	1.00	—	33	1.00	—
54.13–69.20	50	188	1.27	(0.71–2.29)	24	2.05	(0.88–4.80)	15	0.51	(0.22–1.21)
69.21–87.61	55	188	1.40	(0.71–2.75)	26	2.47	(0.94–6.46)	24	0.81	(0.33–2.00)
≥87.62	56	188	1.36	(0.54–3.43)	27	1.97	(0.53–7.28)	23	0.95	(0.27–3.35)
p value for trend			0.923			0.664			0.331	
Saturated fat (g)										
≤14.13	47	188	1.00	—	15	1.00	—	27	1.00	—
14.14–19.40	50	188	2.42	(1.31–4.47)	22	4.45	(1.70–11.62)	20	1.26	(0.56–2.86)
19.41–25.34	58	188	3.18	(1.62–6.21)	32	8.81	(3.15–24.65)	21	1.13	(0.46–2.81)
≥25.35	65	188	4.37	(1.89–10.12)	29	7.53	(2.13–26.59)	27	2.36	(0.76–7.39)
p value for trend			0.002			0.011			0.161	
Mono-unsaturated fat (g)										
≤15.77	47	188	1.00	—	17	1.00	—	25	1.00	—
15.78–21.15	53	188	2.24	(1.21–4.15)	23	2.99	(1.23–7.29)	21	1.24	(0.54–2.85)
21.16–27.89	59	188	2.80	(1.38–5.65)	30	4.22	(1.55–11.47)	23	1.41	(0.55–3.63)
≥27.90	61	188	3.45	(1.39–8.56)	28	3.85	(1.09–13.64)	26	2.58	(0.76–8.75)
p value for trend			0.024			0.072			0.448	
Polyunsaturated fat (g)										
≤18.34	83	188	1.00	—	34	1.00	—	40	1.00	—
18.35–22.46	55	188	0.61	(0.36–1.04)	29	0.83	(0.40–1.73)	19	0.48	(0.22–1.05)
22.47–27.71	44	188	0.26	(0.14–0.48)	17	0.36	(0.15–0.86)	18	0.16	(0.06–0.41)
≥27.72	38	188	0.20	(0.09–0.46)	18	0.24	(0.08–0.78)	18	0.20	(0.06–0.67)
p value for trend			<0.001			<0.001			<0.001	
Cholesterol (mg)										
≤190.50	46	188	1.00	—	21	1.00	—	22	1.00	—
190.51–264.03	47	188	1.58	(0.87–2.87)	22	2.17	(0.95–4.98)	18	0.89	(0.40–2.11)
264.04–359.51	57	188	1.77	(0.96–3.24)	26	1.96	(0.84–4.58)	24	1.17	(0.49–2.78)
≥359.52	70	188	2.39	(1.23–4.64)	29	2.39	(0.95–6.04)	31	1.95	(0.76–4.96)
p value for trend			0.008			0.040			0.136	
Carbohydrates (g)										
≤210.46	58	188	1.00	—	21	1.00	—	28	1.00	—
210.47–269.10	41	188	0.82	(0.45–1.49)	23	1.26	(0.55–2.86)	15	0.66	(0.27–1.60)
269.11–349.09	63	188	1.25	(0.63–2.51)	28	1.55	(0.58–4.06)	26	1.57	(0.60–4.10)
≥349.10	58	188	1.10	(0.40–2.99)	26	1.24	(0.31–5.04)	26	2.45	(0.62–9.59)
p value for trend			0.141			0.157			0.993	
Fiber (g)										
≤18.27	65	188	1.00	—	27	1.00	—	30	1.00	—
18.28–25.28	62	188	0.93	(0.53–1.60)	27	0.92	(0.43–1.95)	27	1.04	(0.48–2.27)
25.29–33.35	58	188	0.56	(0.30–1.08)	30	0.75	(0.32–1.72)	21	0.53	(0.21–1.35)
≥33.36	35	188	0.22	(0.09–0.55)	14	0.16	(0.04–0.59)	17	0.48	(0.13–1.78)
p value for trend			<0.001			<0.001			0.005	

[1]Adjusted by: age (5-year age groups), gender, total calories, chili-pepper consumption, socio-economical status, cigarette smoking, salt consumption, history of peptic ulcer, type of interview (direct *vs.* surrogate), duration of interview, place of interview (hospitals *vs.* home).

TABLE IV – ADJUSTED ODDS RATIOS (OR)[1] FOR THE EFFECT OF MICRONUTRIENT INTAKE ON GASTRIC CANCER

Nutrient/day	All adenocarcinomas				Intestinal (n = 98)			Diffuse (n = 95)		
	Cases	Controls	OR	95% CI	Cases	OR	95% CI	Cases	OR	95% CI
Vitamin A (eq. ret.)										
≤1,742.60	45	188	1.00	—	19	1.00	—	21	1.00	—
1,742.61–2,627.53	73	188	2.10	(1.18–3.74)	36	2.82	(1.27–6.25)	29	2.20	(0.96–5.04)
2,627.54–3,733.60	44	188	1.34	(0.71–2.54)	19	1.50	(0.61–3.67)	20	1.50	(0.60–3.74)
≥3,733.61	58	188	1.89	(0.95–3.77)	24	1.81	(0.68–4.76)	25	2.00	(0.73–5.48)
p value for trend			0.450			0.709			0.995	
Carotene (eq. ret.)										
≤1,004.21	39	188	1.00	—	16	1.00	—	16	1.00	—
1,004.22–1,669.86	68	188	1.77	(0.99–3.17)	30	1.97	(0.87–4.45)	30	2.41	(1.01–5.75)
1,669.87–2,422.91	69	188	1.75	(0.94–3.24)	32	1.79	(0.76–4.23)	31	3.00	(1.18–7.68)
≥2,422.92	44	188	0.75	(0.35–1.61)	20	0.61	(0.21–1.73)	18	1.45	(0.46–4.53)
p value for trend			0.463			0.296			0.882	
Vitamin C (mg)										
≤186.58	77	188	1.00	—	17	1.00	—	17	1.00	—
186.59–279.43	57	188	1.55	(0.87–2.76)	24	1.02	(0.44–2.32)	32	2.13	(1.17–6.39)
279.44–392.03	34	188	1.10	(0.58–2.10)	24	0.12	(0.47–2.70)	21	1.45	(0.54–3.84)
≥392.04	52	188	1.30	(0.62–2.73)	33	0.97	(0.36–2.61)	25	2.65	(0.85–8.25)
p value for trend			0.959			0.380			0.095	
Vitamin E (eq. alphatocopherol)										
≤12.56	39	188	1.00	—	30	1.00	—	37	1.00	—
12.57–15.61	65	188	0.65	(0.38–1.11)	29	1.02	(0.48–2.19)	22	0.51	(0.24–1.10)
15.62–19.54	49	188	0.29	(0.15–0.57)	16	0.59	(0.24–1.43)	14	0.18	(0.06–0.49)
≥19.55	67	188	0.36	(0.16–0.82)	23	0.59	(0.19–1.85)	22	0.34	(0.10–1.17)
p value for trend			<0.001			0.001			0.005	
Folate (μg)										
≤257.40	64	188	1.00	—	26	1.00	—	32	1.00	—
257.41–346.30	54	188	1.15	(0.57–1.99)	25	1.12	(0.52–2.45)	21	0.91	(0.41–2.02)
346.31–466.25	52	188	1.06	(0.57–1.99)	28	1.35	(0.58–3.16)	19	0.80	(0.33–1.97)
≥466.26	50	188	1.00	(0.45–2.27)	19	0.70	(0.22–2.23)	23	1.23	(0.39–3.84)
p value for trend			0.116			0.036			0.535	

[1]Adjusted by: age (5-year age groups), gender, total calories, chili-pepper consumption, socio-economical status, cigarette smoking, salt consumption, history of peptic ulcer, type of interview (direct *vs.* surrogate), duration of interview, place of interview (hospital *vs.* home).

1990; Ramon *et al.,* 1993; Hansson *et al.,* 1994), but only 2 of them showed a significant reduction of gastric-cancer risk (González *et al.,* 1994; Risch *et al.,* 1985). In addition, the consumption of foods rich in fiber has consistently been associated with moderate to strong decreases of gastric cancer (World Cancer Research Fund and American Institute for Cancer Research, 1997). Considering that the results of the studies published to date are inconsistent, and that the potential mechanism by which dietary fiber may protect against gastric cancer remains unidentified, further research should try to rule out the possibility that fiber consumption could simply be an indicator for fruit and vegetable consumption.

The epidemiologic results on vitamin-E consumption and gastric-cancer risk are also inconsistent: 3 case-control studies showed a reduction of about 40% in the risk for gastric cancer, corresponding to the highest level of vitamin E intake (Buiatti *et al.,* 1990; Ramon *et al.,* 1993; Hansson *et al.,* 1994), while others found no such association (World Cancer Research Fund and American Institute for Cancer Research, 1997). In view of the anti-oxidant properties of vitamin E, it has been suggested that this compound may protect against gastric cancer by inhibiting the formation of *N*-nitroso compounds, which have been considered as risk factors for that tumor (Kono and Hirohata, 1996). Our results support the hypothesis that vitamin E might reduce the risk of gastric cancer.

In our study, the highest level of folate consumption showed a protective effect only for cases of the intestinal type of gastric cancer. Freudenheim *et al.* (1991) found a protective effect of high folate consumption for rectal cancer. In spite of the evidence that folate intake may inhibit neoplastic changes in humans, a potential mechanism for reducing gastric-cancer risk is not known. It is possible that fiber, vitamin-E and folate consumption could explain the consistent protective effect of fruit and vegetables; however, the findings are not consistent across published studies of nutrients and gastric cancer.

Information on a potentially protective effect of vegetable—fat consumption (polyunsaturated fat), or regarding specific types of polyunsaturated fatty acids and gastric cancer is very limited. In a study from Spain, González *et al.* (1993) observed a protective effect for gastric cancer in relation to the consumption of borage, a vegetable rich in gamma-linoleic acid, while a study from Italy reported that the consumption of vegetable fat was inversely related to gastric-cancer risk (Buiatti *et al.,* 1990). These findings are consistent with our results of about 80% reduction in the risk for the highest level of polyunsaturated-fat consumption. The main sources of this type of fat in the Mexican diet are sunflower and safflower oils (University of Texas, Houston Health Science Center, School of Public Health, Human Nutrition Center, United States Department of Agriculture, Agricultural Research Service, 1996). Other sources of polyunsaturated fatty acids include vegetable margarines and some kinds of nuts (World Cancer Research Fund and American Institute for Cancer Research, 1997). Future research should focus on the assessment of this relationship, focusing on details concerning GC risk and the consumption of specific types of fatty acids.

In this study, we failed to show any protective effect of vitamin-C (ascorbic-acid) or carotenoid consumption on gastric-cancer incidence. Such an effect has been consistently reported by epidemiologic studies (World Cancer Research Fund and American Institute for Cancer Research, 1997). At least 2 potential explanations should be considered in this regard: the first is that fruit and vegetable consumption is highly prevalent among the Mexican population (Instituto Nacional de la Nutrición Salvador Zubirán, 1995). Hence, only a small proportion of study subjects reported that they did not consume ascorbic acid and/or carotenoids in their diet. If an effect truly exists, the small difference existing between cases and controls in our study may have limited the statistical power to detect such an association.

A second potential explanation has to do with the consumption of chili pepper in Mexico, and have reported that chili-pepper consumption could be an important risk factor for gastric-cancer incidence in the country (López-Carrillo *et al.,* 1994). Fresh chilies consumed in Mexico are an important source of ascorbic acid and carotenoids for our population (University of Texas, Houston Health Science Center, School of Public Health, Human Nutrition Center, United States Department of Agriculture, Agricultural Research Service, 1996), therefore, it was not surprising to find that vitamin-C and carotenoid consumption are actually slightly higher among cases than among controls. In this sense, vitamin-C and carotenoid consumption should be regarded more as proxy indicators of exposure to chili peppers in Mexico than otherwise. Capsaicin is the compound that gives the pungent, hot flavor to chili peppers, and is also a potential carcinogen. Due to the absence of data on the capsaicin content of different chilies, it was not possible to assess any potential interactions between vitamin C and capsaicin levels.

Although this study was the largest effort made to date in Mexico to evaluate the nutritional contents of the foods consumed in regard to GC, the study was limited by incomplete data on nutrients for some foods in the FIAS 3.0 and Mexican food-composition tables. Thus, some nutritional information of local foods had to be added to the dataset of the FIAS dataset, and where a considerable difference was found between the 2 sources, the information was taken from the Mexican tables.

In summary, our results confirm that diet plays an important role in the etiology of gastric cancer. Consumption of polyunsaturated fat, fiber and vitamin E might confer a protective effect against gastric cancer, while an increased risk was observed with the consumption of saturated fat and, cholesterol. Intervention studies are now required to evaluate the reduction of gastric-cancer risk that may stem from the consumption of nutrient supplements.

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